Cardiac reserve, Cardiac work & Oxygen consumption by the heart

ILOs

By the end of this lecture the students will be able to:

1. By the end of this lecture the students will be able to:
2. Describe cardiac reserve with its parameters limitations, and mechanisms
3. Describe cardiac output curve , hypo-effective and hyper-effective hearts and their causes.
4. List the types of work done by the heart
5. Describe mechanical efficiency of the heart
6. Describe the O_2 consumption by the heart
7. Apply the information studied in this section to solve a clinical problem or explain clinical case.

Cardiac Reserve

- ✓ It is the ability of the heart to augment its CO in order to meet the body demands.
- ✓ It equal the difference between basal and maximal cardiac output or work.
- ✓ Cardiac reserve = Maximal cardiac output - basal cardiac output
  = 35 (L/min) - 5 (L/min).
  = 30 (L/min).

N.B:

- ✓ As the venous return increase to the heart, cardiac output increases up to a limit after which the heart will become a limiting factor for increased cardiac output.
- ✓ Normal cardiac output curve show that heart can increase cardiac output up to 13 L/min (that is 2.5 times the normal cardiac output) before plateau is obtained.
- ✓ This mean that to increase cardiac output above 13 L/min, specific way of heart stimulation are needed.

Mechanisms of cardiac reserve :

This happen through increasing the determinants of CO: stroke volume and heart rate.

A) Short acting mechanisms:

Moment to moment increase in CO to meet the increased demands.

1) Increase CO up to 13 L/min (permissive limit):
   a) Heart rate increase:
      - ✓ from basal 60 beat up to 90 beats which is the intrinsic pacemaker frequency.
      - ✓ Happen by inhibition of vagal tone without sympathetic stimulation.
   b) Stroke volume:
      Increased by increased venous return and EDV by heterometric mechanism (Frank -Starling law). Followed by homomeric regulation (with same EDV) if condition prolonged than 2 minutes , SV is increased by decreased ESV.
2) Increase CO above the permissive limit:
   This depend on sympathetic stimulation and circulating catecholamines. It is due to β1 receptor stimulation.
   a) Heart rate reserve mechanism:
      Sympathoadrenal stimulation exert a +ve chronotropic effect and heart rate can increase up to 180 beat per minute.
   b) Stroke volume reserve mechanism:
      Sympathoadrenal stimulation exert a +ve inotropic effect with constant EDV, SV increase by increasing power of contraction and decreasing ESV down to 30 ml.

B) Long term Mechanisms :
Slowly and gradual acting mechanism:

1) <u>Dilation :</u>
- ✓ Depend on frank starling law increasing EDV.
- ✓ Occur in volume over load as aortic incompetence, ventricular septal defect and congestive heart failure.

2) <u>Hypertrophy:</u>
- ✓ Increased number and thickness of cardiac muscles, increasing the thickness of ventricular wall to overcome the excessive resistance.
- ✓ Occur in pressure overload as in hypertension, aortic stenosis.

Limitation of cardiac reserve:
- ✓ Increased CO up to permissive limit (13L/min) has no limitation.
- ✓ Sympathoadrenal stimulation is limited by stores of norepinephrine and responsiveness to β adrenergic receptors.

- ✓ If heart rate increased above 180 beat per minute:
  - ✓ cardiac output is decreased due to reduced ventricular filling and consequently stroke volume.
  - ✓ Increased work of the heart and O_2 consumption while coronary blood flow is decreased.
- ✓ If EDV is excessively increased, overstretch of the wall with extra need for oxygen occur leading to diastolic dysfunctions.
- ✓ If contractility is excessively increased, injury of myocardium may happen as in athlete heart.
- ✓ If hypertrophy exceeded certain limit, decreased blood supply occur as blood vessels doesn't increase in the same extent as muscle hypertrophy occur (disproportionate).

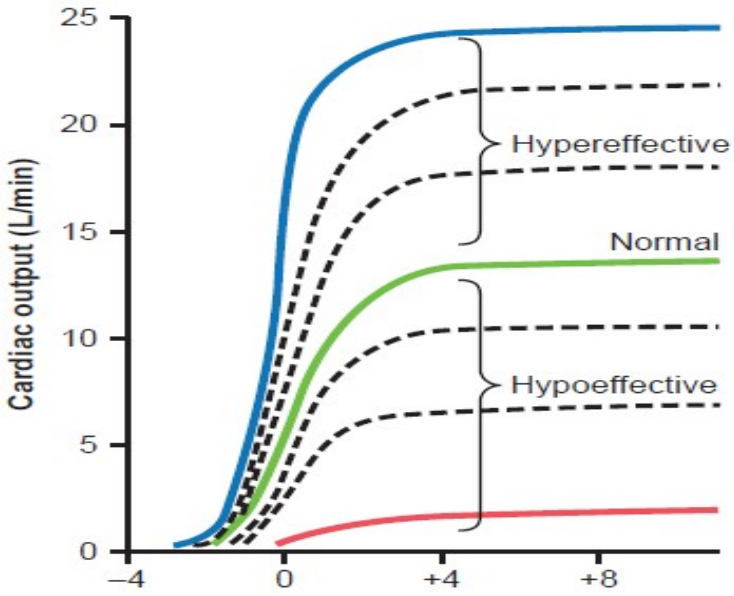
Cardiac output curve:

- ✓ The normal cardiac output curve, showing the cardiac output per minute at each level of right atrial pressure.

- ✓ <u>Causes of hyper-effective heart:</u>
  Hyper-effective heart is heart with increased cardiac output better than normal. It can be caused by:
  1) Nervous stimulation.
     - Nervous excitation through parasympathetic inhibition and sympathetic stimulation, increase heart rate and power of contraction. This can increase cardiac output up to 25 L/min.
  2) Hypertrophy.
     - Work over load for the heart during exercise cause cardiac hypertrophy.
     - In marathon runners, hypertrophy occurs by 50- 75% which leads to increase cardiac output up to 60-100%.

  In marathon runners, both factors are combined so CO can increase up to 30 – 40 L/min.

- ✓ <u>Causes of hypo-effective heart:</u>
  Hypo-effective heart is heart with decreased cardiac output. It can be caused by:
  1) Increased arterial pressure such as in severe hypertension.
  2) Inhibition of nervous excitation of the heart.
  3) Arrhythmias.
  4) Coronary artery insufficiency.
  5) Valvular heart disease.
  6) Congenital heart disease.
  7) Myocarditis.
  8) Cardiac hypoxia.



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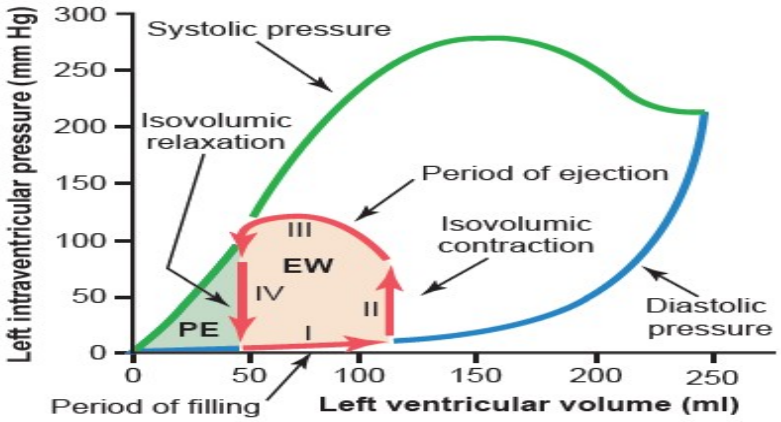
Cardiac work

- ✓ Work is the product of force times distance.
- ✓ In the case of the work done to move a volume of fluid, work is defined as the product of the volume of fluid and the pressure required to move the fluid.

Types of cardiac work:

- ✓ **Stroke Work:**
  - is the product of stroke volume and pressure needed to eject it (equal to mean systolic pressure during ejection).
  - **Stroke work= stroke volume × mean arterial pressure.**
  - Left ventricle have stroke work 7 times than that of right ventricle as aortic pressure is 7 times the pulmonary pressure.
- ✓ **Minute work:**
  - is work done by heart in whole minute.
  - Minute work = stroke work × heart rate.
- ✓ Increasing the pressure with constant stroke volume increase the cardiac work (pressure work).
- ✓ Increasing the cardiac output (stroke volume) with constant pressure increase the cardiac work (volume work).
- ✓ Pressure work consume more O_2 than volume work, so the heart with coronary insufficiency can have angina pectoris more common with aortic stenosis (pressure load) more than with aortic incompetence (volume load).
- ✓ So, work output of heart is in form of
  - External work : include pressure and volume work.
  - Kinetic energy to give velocity to the blood (normally small and neglected = 1%. But increased in case of aortic stenosis).

- And heat.



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Mechanical efficiency of the heart :

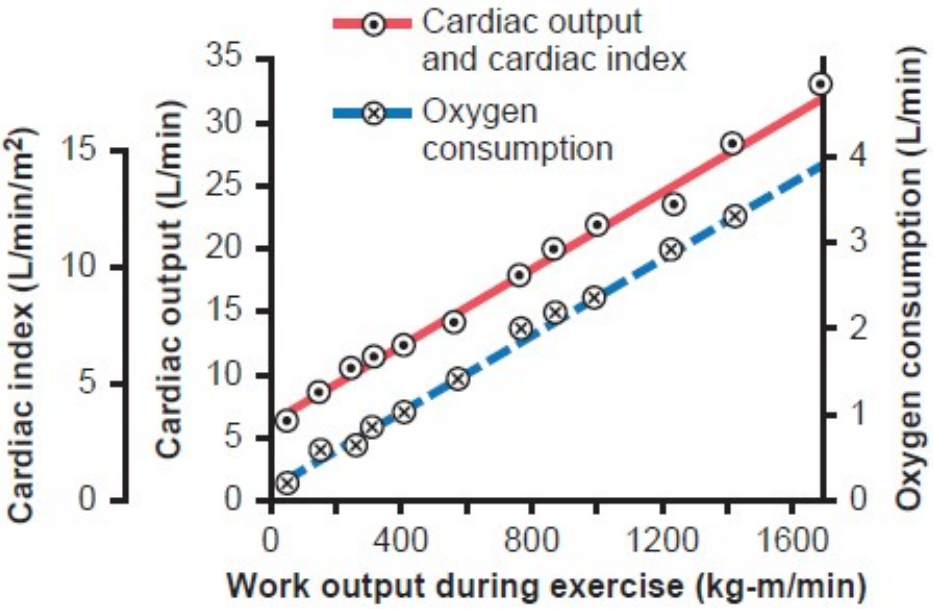
- ✓ The efficiency of the heart may be calculated as the ratio of the work accomplished to the total energy used. Normally, it is 20-25%.
- ✓ It is decreased with increased after load, preload, heart rate and heart dilation.
- ✓ It is increased with hypertrophy.

Oxygen consumption by the heart

- ✓ O_2 consumption by the beating heart is about 9 mL/100 g/min at rest.
- ✓ O_2 consumption increases in exercise and in a number of different states.
- ✓ O_2 extraction by cardiac muscles is high and little additional O_2 can be extracted from the blood in the coronaries. so increases in O_2 consumption require increases in coronary blood flow.

Factors affecting O_2 consumption:

- ✓ O_2 consumption by the heart is determined primarily by the intramyocardial tension, the contractile state of the myocardium, and the heart rate.
- ✓ When the heart rate is increased by sympathetic stimulation, O_2 consumption is increased due to increased number of beats, increased velocity and strength of each contraction.
- ✓ When heart is dilated, according to Laplace law, tension developed in the wall is increased and accordingly O_2 consumption is increased.
- ✓ O_2 consumption is increased with increase in after load and increased preload. But Afterload produce more O_2 consumption.



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